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Intraspinal Seeding from Intracranial Tumors in Children

Philip Stanley[1]
Melvin O. Senac, Jr.
Hervey D. Segall

Cerebrospinal fluid–borne metastases were demonstrated by metrizamide myelography in 20 children with primary intracranial tumors, posterior fossa medulloblastomas being the most common. The lumbosacral region was affected most often, with nerve root thickening, nodularity, and irregularity of the thecal sac. Cord cloaking, nodularity, and meningeal disease were common in the cervical and thoracic areas. Intraspinal spread from primary intracranial tumors signals a poor prognosis. However, the early detection of intraspinal spread afforded by metrizamide myelography can alter therapy and thereby improve prognosis.

Intraspinal spread from primary intracranial tumors by seeding along cerebrospinal fluid (CSF) pathways is well recognized at autopsy, but it is often underdiagnosed during life [1]. However, with the introduction of water-soluble myelographic contrast media, early involvement and the exact extent of the intraspinal spread may be demonstrated [2]. Intraspinal metastatic disease occasions a grave outlook requiring modification of radiation therapy fields and chemotherapeutic regimens [3, 4]. We reviewed the myelographic features of intraspinal deposits in 20 children with histologically proven primary intracranial malignancy.

Materials and Methods

From January 1981 until December 1983, 29 children with histologically proven primary intracranial malignancy had myelograms with water-soluble contrast media to assess intraspinal spread. The 20 children within this group who had radiologic evidence of intraspinal spread form the basis of this report. The ages of these 20 children (11 boys) at the time of initial diagnosis ranged from 1 year, 9 months, to 8 years, 1 month (mean, 4 years, 9 months). In 19 patients, there was histologic confirmation of the primary tumor, and in the other patient, there was a percutaneous biopsy of a lumbar nerve root nodule. Twelve patients had posterior fossa medulloblastomas and three had ependymomas. Undifferentiated small cell malignant tumor, retinoblastoma, lymphoma, brainstem astrocytoma, and a thalamic neuroblastoma were the other primary intracranial tumors. (Nine children with negative myelograms had the following primary intracranial tumors: four medulloblastomas, two ependymomas, one brainstem glioma, one thalamic glioma, and one pinealoma.)

The patients with intraspinal involvement are divided into two basic groups. In the beginning of this series only those patients with intracranial progression of the primary tumor, often with clinical features of spinal involvement and positive CSF cytology, were examined. This group of nine children had myelograms 56–706 days after intracranial surgery (mean, 316 days). The other 11 children who were seen more recently in the series all had myelograms within 3 weeks of posterior fossa surgery (mean, 16 days) irrespective of clinical progression, spinal symptomatology, or CSF changes. This is a reflection of a more aggressive therapeutic approach together with a realization that early subarachnoid spread may be easily detected with water-soluble myelography.

Routine prone and supine myelography using the lumbar route was performed in all 20 patients with metrizamide (220 mg I/ml concentration). The volume of contrast material was determined by the patient's weight. In four patients, spinal computed tomography (CT) was

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[1] All authors: Department of Radiology, University of Southern California, School of Medicine, and Childrens Hospital of Los Angeles, P.O. Box 54700, Los Angeles, CA 90054. Address reprint requests to P. Stanley at Children's Hospital.

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undertaken after metrizamide myelography. Seven patients had repeat myelograms to assess progress of the intraspinal disease.

Results

The distribution of intraspinal spread in this series of 20 patients is shown in figure 1. There was involvement of the lumbosacral region in every patient, and in eight patients this was the sole site of the disease. In three patients, there was very extensive continuous intraspinal spread from the high cervical region to the tip of the thecal sac. There was minimal involvement in three of the 11 patients investigated within 3 weeks of surgery. However, in the other eight patients in this group, involvement was apparent at many levels. In the other group who had myelograms many weeks after intracranial surgery, involvement was confined to the lumbosacral region in three patients; otherwise, several sites were involved.

The radiologic pattern of intraspinal spread was dependent on the site of involvement. Nodularity and irregularity of the

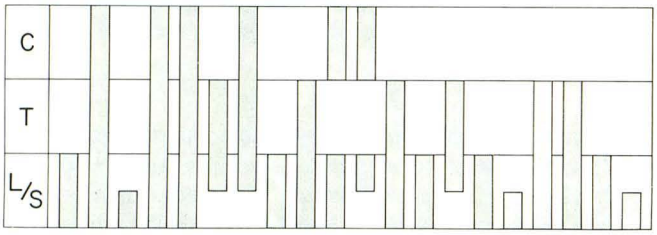

Fig. 1.—Distribution of intraspinal spread in 20 children with primary intracranial malignancy. Each bar represents one patient. C = cervical; T = thoracic; L/S = lumbosacral.

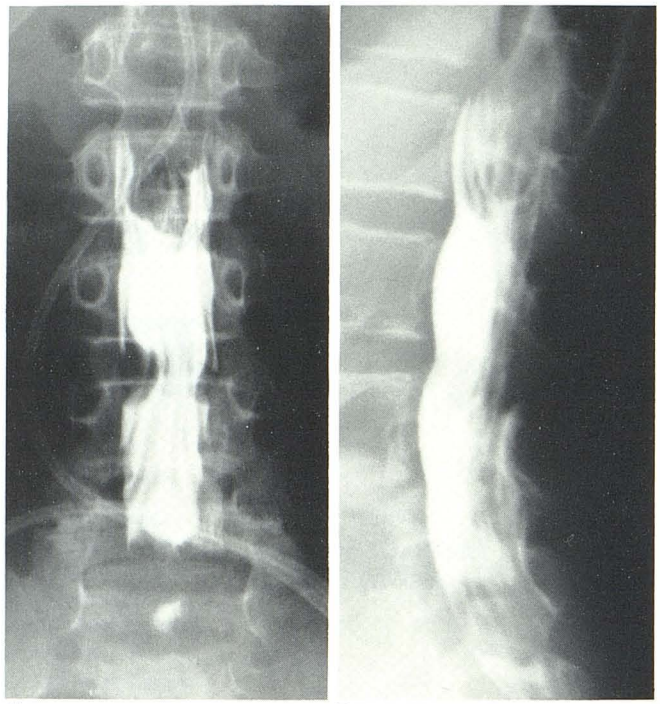

Fig. 4.—Posteroanterior (**A**) and lateral (**B**) projections of prone lumbar myelogram in 3-year-old with intraspinal spread from posterior fossa medulloblastoma. Multiple large intradural masses with thickening of lumbar nerves. High-grade obstruction to cranial flow of metrizamide from most superior mass was also present.

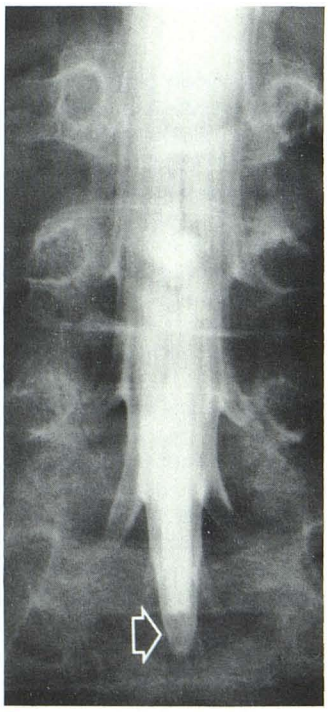

Fig. 2.—Myelogram of 8-year-old boy with posterior fossa medulloblastoma. Obliteration of tip of distal thecal sac by tumor (*arrow*) was unchanged on myelogram 2 months later.

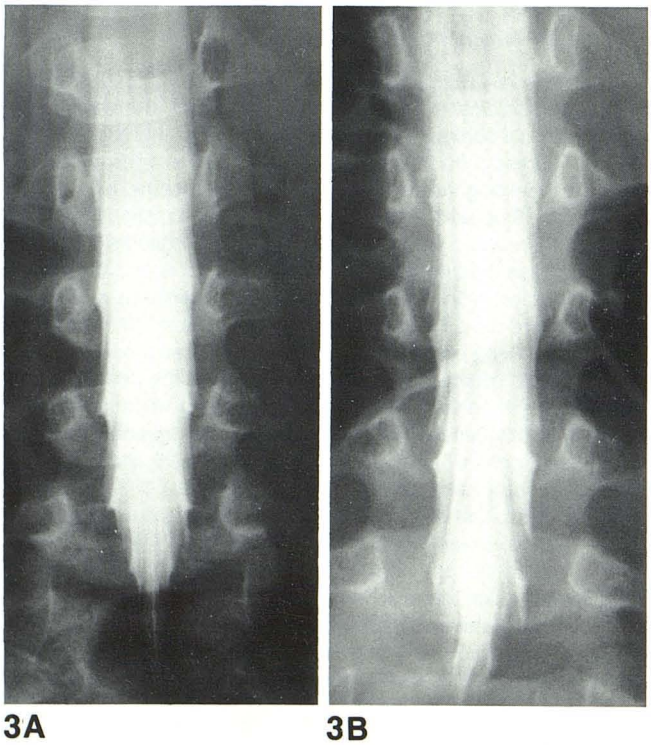

Fig. 3.—3½-year-old boy with intracranial lymphoma and progressive leg weakness. **A**, Initial prone myelogram. Minimal symmetric thickening of lower lumbar and sacral roots. **B**, 2 weeks later. Multiple nerve root nodules with thickening of roots and asymmetry of thecal sac. (Malignant lymphoma cells were present in the CSF.)

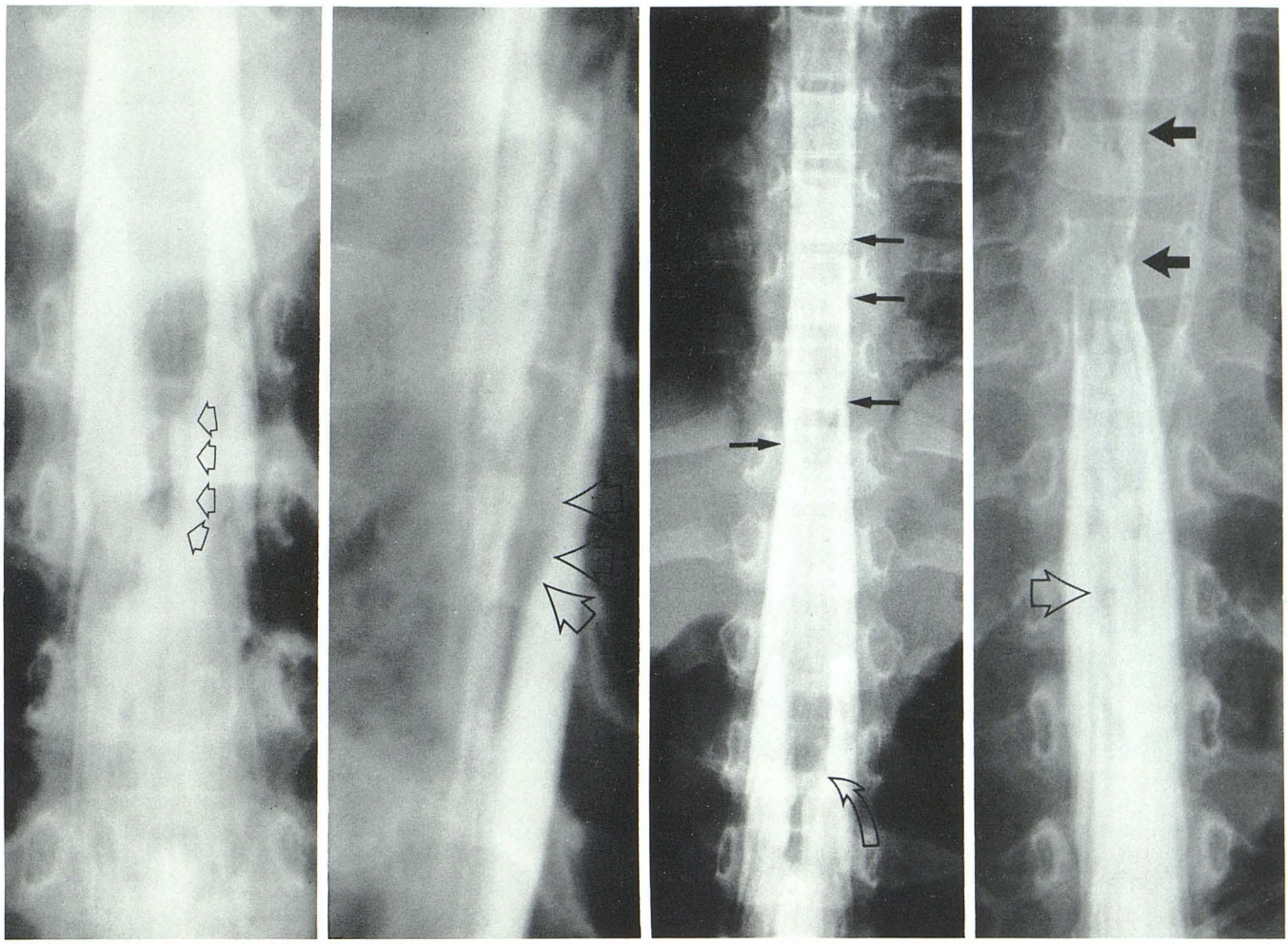

Fig. 5.—3¼-year-old girl with posterior fossa medulloblastoma. **A**, Supine myelogram. Subtle irregularity of conus from tumor seeding (*arrows*). **B**, Lateral view. Irregularity of dorsal surface of conus (*arrows*).

Fig. 6.—Fine irregularity of arachnoid covering thoracic cord (*solid arrows*). Asymmetric thickening of conus, particularly on left side (*curved arrow*).

Fig. 7.—Supine myelogram in 3-year-old girl with poorly differentiated cerebellopontine angle tumor. Irregular extradural mass centered at D8 (*solid arrows*). In addition, there is nodule on conus (*open arrow*).

most caudal aspect of the thecal sac was present in 16 patients. In three patients, this was the only abnormality (fig. 2). Lumbar nerve root nodularity and thickening, usually with obliteration of the accompanying root sleeve, was present in 17 patients. In one patient, progressive involvement of the nerve roots was observed over 2 weeks (fig. 3). In addition to nerve root involvement, large masses in the lumbar region obstructing the cranial flow of metrizamide were seen in one patient (fig. 4). CT after metrizamide myelography in three patients with lumbar nerve root involvement added no new information. Irregular tumor plaques on the conus were present in four patients (fig. 5). Three further patients had nodules on the conus.

In 10 patients, the thoracic cord and surrounding membranes exhibited a variety of patterns of involvement. In three patients, extensive cloaking of the surface of the cord was seen that was so extensive in one as to block the cephalad passage of metrizamide. Multiple discrete nodules on the surface of the cord, particularly its dorsal aspect, were present in five patients. Diffuse arachnoidal irregularity (fig. 6) was present in three patients. In three patients, extradural masses were demonstrated on myelography (fig. 7).

Discrete nodularity on the surface of the cord was present in three of the six patients with myelographic evidence of cervical cord involvement. In two patients, there was extensive cloaking of the cord, with a block to the cranial flow of metrizamide in one patient (fig. 8A). Further information was gained with CT after the myelogram in this patient (figs. 8B and 8C). A lobulated mass projecting from the foramen magnum into the upper cervical subarachnoid space was seen in one patient (fig. 9) who also had the discrete nodule on the dorsal surface of the lower cervical cord.

Six patients who had follow-up myelograms showed progression of the disease (fig. 3). As of December 1983, 12 of the 20 patients had died, with clinical and radiologic evidence of progression of the disease with intracranial manifestations being dominant. Only one of the 12 patients had an autopsy; it confirmed extensive spinal subarachnoid spread. All eight survivors belonged to the group investigated in the immediate postoperative period after posterior fossa craniotomy. How-

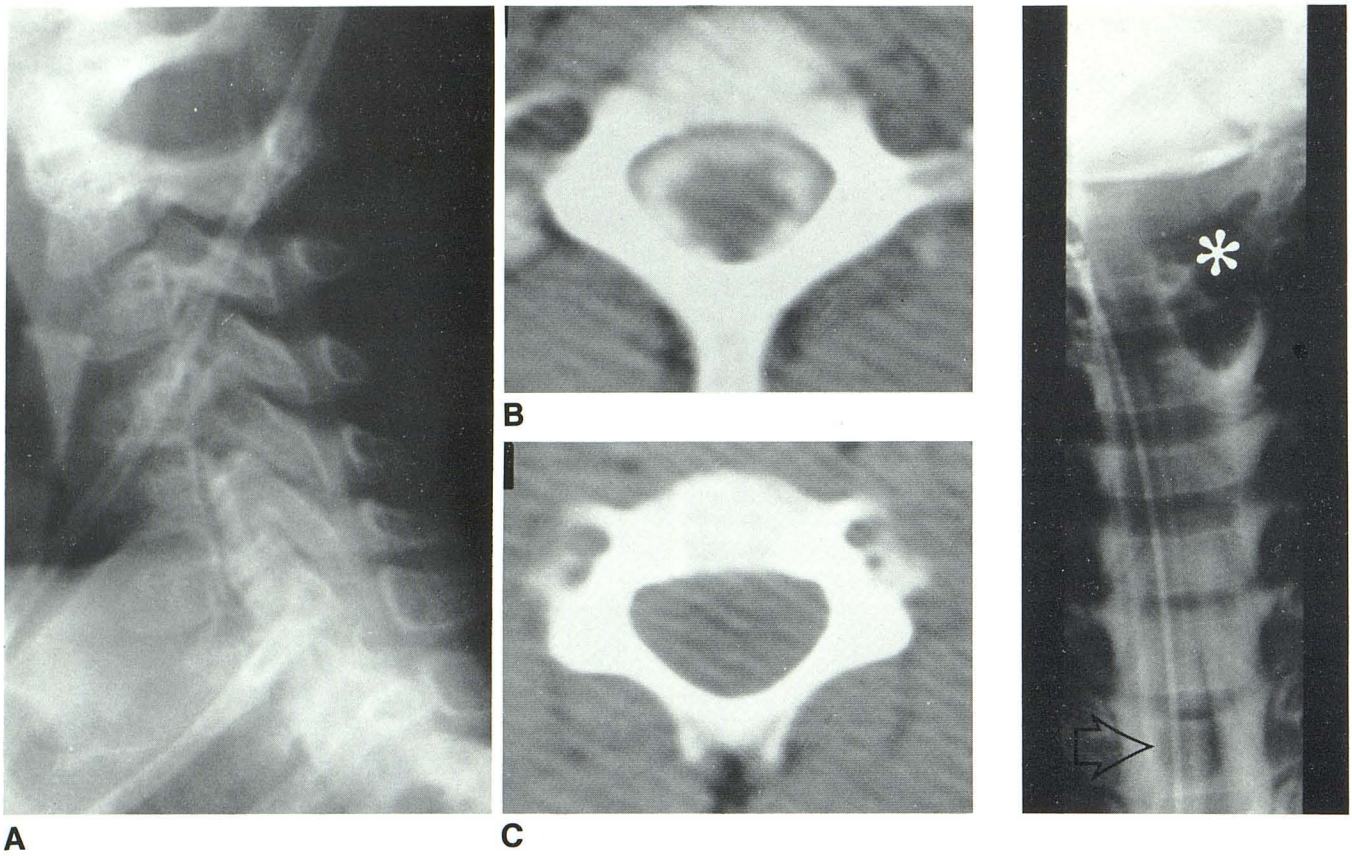

Fig. 8.—3½-year-old girl with subtotal removal of medulloblastoma. **A**, Lateral myelogram. Expansion of cord with complete obstruction of cranial flow of metrizamide. **B**, CT scan at C6–C7. Nodularity on cord surface. **C**, At C4. Complete obliteration of subarachnoid space from extensive cloaking of surface of cord with tumor.

Fig. 9.—Lobulated mass (*asterisk*) in high cervical subarachnoid space continuous with posterior fossa ependymoma in 1¾-year-old girl. Nodule on dorsal surface of cord at C7 (*open arrow*).

ever, one of the eight survivors had an uncontrollable recurrence of the primary tumor and a second patient had progressive spinal subarachnoid disease.

Discussion

The occurrence of metastases from intracranial tumors by way of seeding along CSF pathways both within the cranial cavity and spinal canal is a well recognized pathologic entity. Medulloblastoma is the childhood brain tumor that most often gives rise to intraspinal implants [5]. In this myelographic study, 12 of 16 children with medulloblastomas had radiologic evidence of spinal involvement. This high frequency and the associated poor prognosis have been reported [3, 6, 7]. Ependymoma is the second most common tumor to seed [8] (three of five patients in our series) followed by pineal neoplasms, astrocytomas, lymphomas, choroid plexus papillomas, and retinoblastomas [5, 9, 10]. This type of metastatic spread has not been diagnosed accurately until the advent of water-soluble contrast myelography. Although Wood et al. [11] concluded that Pantopaque (iophendylate) was satisfactory in detecting and localizing spinal cord metastasis, Bryan [1] found visible nodules after Pantopaque myelography in only one of 22 autopsy-proven cases of spinal metastases from CSF seeding. With metrizamide, subtle abnormalities may be detected that would have been obscured with high-density Pantopaque. The use of metrizamide with routine myelography after posterior fossa surgery accounts for the increased detection of spinal subarachnoid spread, which has been reported recently, especially with medulloblastomas [2].

A variety of patterns of intraspinal spread are seen. In the lumbosacral region nodularity and irregularity of the distal sac is common, as is nodularity and thickening of the nerve roots with obliteration of the root sleeves. The high incidence of involvement of the lumbosacral regions shows the effect of gravity on the CSF-borne metastasis. Diffuse thickening with adhesions of the nerve roots and irregular obliteration of the subarachnoid space may resemble chemical or postinfective arachnoiditis. Symmetric thickening of the nerve roots may also simulate hypertrophic neuritis, a rare occurrence in children [12]. In the one patient who had an autopsy and a second patient who had a percutaneous biopsy, histologic

examination confirmed that the root thickening was from tumor invasion.

In the cervical and thoracic regions, a variety of appearances are seen, reflecting involvement of the cord and surrounding membranes. Nodularity or cloaking of the cord with plaques of tumor is common, a finding often observed at autopsy. Apparent widening of the cord is almost certainly from extensive plaques and not from intramedullary metastasis, which is rare. However, metastatic disease may invade the cord parenchyma from the surface [5], a feature that is not possible to assess accurately in radiologic studies. The plaques and nodularity were more common in the dorsal region, supporting the theory of spread along CSF pathways, the normal caudad flow of CSF being along the dorsal aspect of the cord [13]. There was, in addition, fine irregularity of the arachnoid, reflecting diffuse meningeal involvement, and extradural disease was found, but less often. In one patient with intraspinal involvement, there was a lobulated mass in the high cervical subarachnoid space continuous with the primary ependymoma in the posterior fossa. This association has been reported [8].

Several points relating to the myelographic technique need emphasizing. It is important that all air bubbles are meticulously excluded from the contrast medium as they can mimic tumor nodules. A technically inadequate puncture, with the needle tip only partially within the subarachnoid, can also mimic malignancy. Because there is a tumor predilection for the dorsal surface of the cord, supine myelography is essential. The detail provided by metrizamide is sufficient, so CT is rarely required, but it was helpful in one patient with apparent widening of the cord.

Ependymal breaching by the primary intracranial tumor or fissuring secondary to hydrocephalus is a source of subarachnoid seeding [5, 14]. Fragmentation of a tumor bathed with CSF, possibly precipitated by trauma, is another important factor [15]. It has been suggested that tumor debris released at the time of the initial craniotomy may be responsible for seeding [5]. Whereas this may be a factor, postmortem demonstration of well established spinal subarachnoid disease has been demonstrated in children dying within days after posterior fossa surgery [5]. Also, subarachnoid spread has been seen at the time of original craniotomy [16–18]. In addition, eight of the 11 children in this series examined within 3 weeks of craniotomy showed extensive disease, indicative that spread was present before the initial surgery.

Reports of improved prognosis in patients with intraspinal deposits from medulloblastomas have given credit to early diagnosis and vigorous therapy to the spine, particularly with radiation [6]. Although this improvement was not reflected in this series of patients, many of whom had advanced disease, the importance of detecting early intraspinal involvement cannot be overemphasized [2]. Metrizamide myelography will provide this information. We maintain that all children with posterior fossa medulloblastomas or ependymomas should have routine postoperative myelography, even though they have no spinal symptoms. In children with other tumors known to spread to the spinal subarachnoid space, myelography should also be performed, particularly if there is clinical or cytologic evidence of spinal subarachnoid involvement or there is failure to control the primary intracranial lesion.

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